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Effects of Thoracic Spine Position during Cycle Sprint Recovery

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By

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Masters of Science

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MASTER'S THESIS

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Deanna Emnott

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Abstract

There is a paucity of research on how to recover during a race or practice immediately between cycling sprints. The subjects of this study included 13 competitive male cyclists recruited from local bicycle shops. This study utilized a pretest-posttest experimental design. Participants completed two 30-s maximal effort sprints on a cycle ergometer followed by two four-min active recovery intervals. They were randomly assigned to either a flexed thoracic spine position greater than 14° (FC) or a neutral thoracic spine position (NC) during cycling sprint recovery intervals on the first testing day and completed the other no less than 48 hours later. Recorded variables included heart rate recovery (HRR), tidal volume (V_T), carbon dioxide output (VCO_2), change in sprint mean power (ΔMP), and change in sprint fatigue index (ΔFI). There were no significant differences between conditions in any of the variables (p>0.05). Using the Cohen's d statistic, there was a small effect of thoracic spine position during recovery on HRR (p=0.293; d=0.33), V_T (p=0.121; d=0.34), and ΔFI (p=0.289; d=0.45) from one sprint to another. However, there was no effect of thoracic position on VCO_2 (p=0.794; d=0.062) or the ΔMP (p=0.853; d=0.051) from sprint to sprint. HRR was 23.5±0.40 bpm in FC and 21.3±5.0 bpm in NC. V_T was 3.0±0.51 L in FC and 3.19±0.54 L in NC. VCO_2 was 3.28±0.25 L/min in FC and 3.26±3.61 L/min in NC. ΔMP was -29.7±17 W in FC and -28.8±19 W in NC. ΔFI was 0.59±3.6 W/s in FC and -0.429±1.9 L in NC. There may be little to no benefit in assuming a more flexed thoracic position between cycling sprints.

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Chapter I

The Problem and Its Scope

Introduction

The autonomic nervous system (ANS) and respiratory system play an integral part in exercise performance and recovery. Recovery is dependent on the reactivation of the parasympathetic nervous system (PNS) after exercise. Cyclists have to recover between sprints during competition and training. During cycling sprints, the sympathetic nervous system (SNS) branch of the ANS becomes more activated and the PNS becomes less activated. During recovery after exercise, the PNS is reactivated. The heart rate (HR) and the beat-to-beat variability in heart rate return back to resting levels (Guerra et al., 2014). The volume of oxygen consumed (VO_2) and tidal volume (V_T) are increased by cycling sprints and then decrease back to a resting state post-exercise (Carter, Dekerle, Brickley, & Williams, 2005).

Heart rate recovery (HRR) is used as a measurement of the parasympathetic reactivation and sympathetic withdrawal following exercise (Borresen & Lambert, 2012; Pierpoint & Voth, 2004; Savin, Davidson, & Haskell, 1982; Shetler et al., 2001). HRR is defined as the change in HR immediately after exercise until one minute post (Javorka, Zila, Balhárek, & Javorka, 2002). The increase in HR during exercise and decrease in HR after exercise is one of the variables modulated by the autonomic nervous system in efforts to maintain sympathovagal balance. The reactivation of the PNS can also be measured by heart rate variability (HRV) and is dependent on body position (Barak et al., 2010). HRV is the beat-to-beat changes in the duration of the cardiac

cycle (Levy et al., 1998). However, the measurement of HRV is most accurate in a completely rested state and supine position (Javorka, Zila, Balhárak, & Javorka, 2002).

Studies have shown that parasympathetic reactivation can be modulated by the respiratory system (Pöyhönen et al., 2004; Yasuma and Hayano, 2004). Tidal volume can affect sinus arrhythmia (RSA) (Yamamoto, Miyachi, Saitoh, Saitoh, & Saitoh, 2001). Tidal volume may also affect carbon dioxide production (VCO_2). In a study on 13 dogs, a doubling of V_T resulted in an average increase in VCO_2 of about 35% (Slutsky et al., 1981). VCO_2 is also altered by training status and aerobic fitness. Multiple sprint-type sports games players have a higher VCO_2 than endurance-trained runners during post-exercise recovery (Hamilton, Nevill, Brooks, & Williams, 1991). This may be due to greater increases in buffering capacity within the muscle, allowing for a higher potential for glycolytic enzyme activity (Parkhouse & McKenzie, 1984; Sharp et al., 1986). This may explain why multiple-sprint-type sports players also showed higher mean anaerobic power output (MP) than endurance-trained athletes (719 and 657 Watts, respectively) (Hamilton, Nevill, Brooks, & Williams, 1991). The Wingate test (WT) is a reliable method for testing MP with a test-retest coefficient where $r > 0.91 - 0.93$ (Bar-Or, 1987; Neptune & Kautz, 2001; Patton, Murphy, & Frederick, 1985). In addition to MP, fatigue index (FI) is another variable that can be used to measure performance. FI determines the rate at which a subject fatigues during a WT (Spierer et al., 2004; Lopez, Smoliga, & Zavorsky, 2014).

The Wingate test is a standardized procedure to measure athletic performance. Posture is an easy variable to manipulate between WTs and may play a role in how well an athlete performs. The thoracic spine includes the vertebrae from T1 to T12 and connects to the ribs. Additional thoracic flexion may increase the zone of apposition (ZOA) and more thoracic extension may decrease it (Lee, 1993). The ZOA is a term used to describe the mechanical

coupling of the diaphragm and the ribcage (Boynton, Barnas, Dadmun, & Fredberg, 1991; Mead, 1979). An optimal ZOA may maximize the contraction of the diaphragm (Lando et al., 1999). V_T is a variable used to measure the depth of one breath (Pöyhönen, Syväoja, Hartikainen, Ruokonen, & Takala, 2004). V_T may also be increased with thoracic flexion. A higher V_T during recovery could be beneficial to cyclists during a maximal sprint. Knowing whether or not thoracic flexion changes recovery variables and impacts subsequent performance may give insight on how to recover for optimal autonomic adaptation and respiratory function.

Purpose of the study

The current study was conducted to determine the effect of thoracic spine position on maximal sprint cycling exercise recovery and possible impacts on subsequent performance. Competitive cyclists preformed two maximal Wingate Anaerobic Tests on a cycle ergometer. After each WT there was a four-minute active recovery period pedaling at a light intensity. During both active recovery intervals, each cyclist acted as their own control and maintained one of two spinal postures. Each subject recovered with a neutral thoracic spine and a more flexed thoracic spine ($\geq 14°$) during separate sessions. Thoracic angle was measured every 30 seconds during each recovery period with an inclinometer to ensure flexed and neutral positions remained consistent throughout recovery. There was at least 48-hours between sessions during which subjects were asked to refrain from any heavy eccentric, unusual exercise, or high intensity cycling bouts.

Null hypothesis

The null hypothesis states that there is no effect of thoracic spinal position on heart rate recovery (HRR), carbon dioxide production (VCO_2), and tidal volume (V_T) during maximal

sprint cycling exercise recovery. The null hypothesis also states that there is no effect of thoracic position on mean power (MP) or fatigue index (FI) during repeat Wingate tests.

Significance of the study

Improving respiration and autonomic profile during recovery may improve performance. The direct effect of thoracic position on recovery has not yet been studied. More thoracic flexion may improve the ZOA and thus the efficiency of the diaphragm (Lee, 1993; Lando et al., 1999). More thoracic flexion has also been correlated with greater V_T (Paek, Kelly, & McCool, 1990). Better HRR has been associated improvements in cycling performance (Lamberts, Swart, Noakes, & Lambert, 2011). It may be beneficial for cyclists, coaches and trainers to know how different positions of the thoracic spine during recovery can affect physiological variables.

Limitations of the study

1. The study took place in the Biomechanics Laboratory and an ergometer was used instead of a regular bike, thus decreasing external validity.
2. It is assumed that subjects tried equally hard on each test. To the best ability of the proctor, an equal amount of encouragement was given to each subject.
3. Subjects are all competitive cyclists, who had completed at least one race within the 12 months prior to this study, however the amount and type of cycling experience varied.
4. Participants may have a preference for cycling recovery posture that they find comfortable and adjusting that may change motivation.
5. Conditions were randomized, however data may be affected by participant familiarity to the sprinting protocol.

Definition of terms

Carbon dioxide production (VCO_2): The volume of carbon dioxide output per unit of time (Smolka, Borkowski, & Zaton, 2014).

Electrocardiogram (ECG): A continuous recording of the electrical changes from myocardium depolarizations during the cardiac cycle (Task Force of The European Society of Cardiology and The North American Society of Pacing and Electrophysiology, 1996).

Fatigue Index (FI): The rate at which fatigue occurs. Calculated as [(max power – min power)/duration]

Heart rate recovery (HRR): The change in HR immediately after exercise until one minute post (Javorka, Zila, Balhárek, & Javorka, 2002).

Heart rate variability (HRV): The variation in the time period between heartbeats (Levy et al., 1998).

Mean anaerobic power (MP): The average power produced during the Wingate test (Koutedakis, Ridgeon, Sharp, & Boreham, 1993).

R-R interval (R-R): The time interval, in milliseconds, between two heartbeats (Levy et al., 1998).

Respiratory sinus arrhythmia (RSA): HRV in synchrony with respiration, by which the R-R interval on an ECG is shortened during inspiration and prolonged during expiration (Yasuma & Hayano, 2004).

Sympathetic autonomic nervous system (S-ANS): The autonomic nervous system branch that increases heart rate, vasoconstriction and blood pressure (Eckberg, Nerhed, & Wallin, 1985).

Parasympathetic autonomic nervous system (P-ANS): The autonomic nervous system branch that decreases heart rate and increases intestinal activity (Eckberg, Nerhed, & Wallin, 1985)

Sympathovagal balance: The balance between the effects of the sympathetic and parasympathetic autonomic nervous systems (Levy et al., 1998).

Thoracic flexion (TF): The angle at which the thoracic spine (from T1 to T12) flexes anteriorly (Hajibozorgi and Arjmand, 2016).

Tidal volume (V_T): The depth of one breath (Pöyhönen, Syväoja, Hartikainen, Ruokonen, & Takala, 2004).

Wingate Anaerobic Test: A 30-second all out exhaustive power test on an ergometer where the subject pedals at a resistance relative to their body weight (Zupan et al., 2009).

Zone of Apposition (ZOA): The structural connection of the diaphragm to the ribcage (Boynton, Barnas, Dadmun, & Fredberg, 1991; Mead, 1979).

Chapter II

Review of Literature

Introduction

The purpose of this review is to examine the effects of cycling sprint recovery spinal posture, specifically thoracic position, on recovery and subsequent cycling performance. The reactivation of the parasympathetic nervous system (PNS) and the deactivation of the sympathetic nervous system (SNS) after exercise can be measured by HRR and HRV (Javorka, Zila, Balhárak & Javorka, 2002; Shetler et al., 2001) and is discussed in greater detail. Recovery variables included in this study were heart rate recovery (HRR), carbon dioxide production (VCO_2), and tidal volume (V_T). This review also examines the use for mean anaerobic power (MP) and fatigue index (FI) in determining how subsequent performance may be affected by recovery. Faster parasympathetic reactivation is a sign of good cardiovascular fitness (Daane, Lamberts, Kallen, Jin, & Van Meeteren, 2012; Guerra et al., 2014).

This review aims to explore how the autonomic nervous and respiratory systems are affected by exercise and spinal position, as well as describe the literature that relates thoracic spine position to exercise recovery and MP. There appears to be an effect of abdominal verses thoracic expansion on V_T (Ohashi, Kamioka, & Matsuoka, 2001). A connection between the ANS and the respiratory system also exists (Yasuma & Hayano, 2004). There was a reported increase in the high frequency (HF) component of HRV with greater V_T (Pöyhönen, Syväoja, Hartikainen, Ruokonen, & Takala, 2004). Also, another study demonstrated that V_T increased

with spinal flexion (Paek, Kelly, & McCool, 1990). The variables that affect recovery and performance resulting from spinal position changes are explored in this review.

Review of Pertinent Literature

Interventions to Enhance Recovery Between Cycling Sprints. Athletic performance may be improved by enhancing recovery from training and competition. Utilizing appropriate strategies for recovery is believed to enhance performance and minimize injury risk (Fridén, Sjöström. & Ekblom, 1981; Mujika, 2012; Otter, Brink, van der Does, & Lemmink, 2016). In some competitive events, athletes are required to repeat maximal bouts of exercise with less than 30 minutes of recovery between bouts. In the London Olympics, Women's Keirin athletes were given 30 minutes between the end of the first round and the beginning of the repechage. Then, they had 45 minutes of recovery between the first and second rounds of repechage (Argus, Driller, Ebert, Martin, & Halson, 2013).

There is a growing amount of research now dedicated to investigating methods to improve recovery from exercise, such as increasing blood flow and airway clearance post-exercise. Recovery between cycling sprints is an important factor, because it affects subsequent performance. Duration of the recovery period has had an effect on cycling performance. Peak power output was 16.1±4.2% lower (p<0.001) with 30 s compared to 180 s of recovery (Monks et al., 2016). Active recovery resulted in a higher mean power than passive recovery (Hedges's g=0.50, p<0.01) (Lopez, Smoliga, & Zavorsky, 2014).

Compression garments have been used for a faster recovery (Argus, Driller, Ebert, Martin, & Halson, 2013). Electrical muscle stimulation has been used to increase blood flow, thus aiding in recovery (Grunovas, Silinskas, Poderys, & Trinkunas, 2007). Also, humidification

therapy has been used to improve airway clearance (Basner, 2007). In a study done comparing the effectiveness of compression garments, electrical muscle stimulation, and humidification therapy, highly trained cyclists performed three bouts of maximal cycling sprints with 30 min recovery periods. The humidification therapy recovery had the best percentage change in mean 30 s power output between sprints two and three compared to passive recovery (2.2 ± 2.5%). Second best was the compression garment recovery that improved by 1.2 ± 1.9% compared to passive recovery. The electrical muscle stimulation group decreased in average power output compared to the passive recovery group by –0.6 ± 2.7% (Argus, Driller, Ebert, Martin, & Halson, 2013). These studies tend to focus on recovery techniques that can be used only after the race or training session is over. However, it may be more functional for an athlete or coach to know the best way to recover between bouts of maximal effort during training or competition.

Parasympathetic Reactivation. During exercise, cardiac output increases as a result of cardiac pumping autoregulation in response of the SNS to a higher metabolic demand. The PNS then reduces cardiac output following exercise (Javorka, Zila, Balhárak & Javorka, 2002). HRR is the rate at which heart rate (HR) declines and is usually measured within minutes following a bout of exercise (Borresen & Lambert, 2007; Lamberts et al., 2008; Shetler et al., 2001). HRR improves with training (Daanen et al., 2012). The ANS regulates both the increase in HR during exercise and the decrease in HR within minutes of the cessation of physical exercise. HRR is characterized by the reactivation of the PNS and the withdrawal of the SNS (Borresen & Lamberts, 2007; Pierpont & Voth, 2004; Savin, Davidson, & Haskell, 1982; Shetler et al., 2001).

HRV and HRR are both measures of parasympathetic reactivation. Studies have failed to find a relationship between HRV and HRR (Buchheit and Gindre, 2006; Javorka, Zila, Balhárek, & Javorka, 2003). These variables may measure different aspects of cardiac parasympathetic

function (Buchheit et al., 2006; Dewland et al., 2007). Heart rate kinetics during the first minute of exercise recovery can be used as an index for autonomic cardiovascular control. Henríquez et al. (2012) studied Brazilian jiu-jitsu wrestlers by recording HRR and HRV as the standard deviation of the normal to normal RR interval (SDNN) following a treadmill test to voluntary exhaustion. HRR and SDNN (in the last 30 seconds of the 1 min recovery interval) were both significantly different when comparing moderately trained (MT) and highly trained (HT) wrestlers. HRR in the first 60 seconds of recovery was about 50.7 bpm for MT and 64.6 bpm for HT. SDNN only showed significant differences between MT and HT in the last 30 seconds of data collection. The standard deviation 45-60 s post-exercise was 9.2 ms for HT and 2.5 ms for MT wrestlers. The differences between training levels may indicate that temporal and non-linear analysis for HR can be used to determine autonomic cardiac control in athletes (Henríquez et al., 2012).

HRR improves with training, even in already well-trained cyclists. Lamberts, Swart, Noakes and Lambert (2008) put trained cyclists with an average VO_{2max} of about 60 ml $kg^{-1}min^{-1}$ and peak power output of 5.2 W kg^{-1} through a high-intensity training (HIT) program. Peak power output in a 40-km time trial improved by 4.7%, 2.2% and HRR improved by 7±6 beats. HRR average was initially 29±6 beats and 35±4 beats post-training. Significant changes were not found in VO_{2max}. The experimenters found that HRR after the 40-km time trial correlated well with improvements in performance variables such as peak power output ($r = 0.73$; $p < 0.0001$) and 40-km time trial ($r = 0.96$; $p < 0.0001$). VO_{2max} appeared to be of limited value with predicting performance.

Lamberts et al. (2010) tested the same cyclists that went through the HIT from the previous study. The experimenters examined the association between training-induced fatigue

and HRR. The purpose was to analyze the relationship between HRR and performance. The experiment split the athletes into a group that continuously increased HRR during the HIT program (1±1 beats) and another group that had two consecutive decreases in HRR during the HIT training period. The group of cyclists that experienced a faster HRR showed a significantly higher MP improvement in the 40-km time trial (20 W) compared with the cyclists that had a slower HRR after HIT training (10 W). There were also more improvements in 40-km time for the group that increased HRR that were not statistically significant. The data suggests that a decrease in HRR is associated with a decrease in endurance capacity for cyclists. Decreased HRR could potentially predict poor performance (Lamberts et al., 2010).

Spine Position. Cycling involves sitting on a bicycle with the trunk flexed so that the cyclist can reach the handlebars. Depending on the level of the athlete, this slumped posture is sometimes maintained for several hours per day. Studies have even found that chronic spinal adaptations occur in master and elite cyclists. Thoracic kyphosis is developed in the standing position (Muyor, López-Miñarro, & Alacid, 2011; Rajabi et al., 2000). Muyor, López-Miñarro, and Alacid (2011) found that the average elite cyclist had a standing thoracic curvature of 48.17±8.05° and the average master cyclist had a thoracic curvature of 47.02±9.24°. Acutely, slumped sitting is associated with changes in respiratory measures. Contrary to the present hypothesis, a slumped seated posture has been correlated with a decrease in expiratory flow, lung capacity, V_T, and breathing frequency (Landers et al., 2003; Lin et al., 2006).

Measurement of the thoracic spine range of motion (ROM) differs somewhat in the research literature. A recent study measured forty healthy young male subjects for thoracic ROM. Subjects were free from any hip, knee or back complications. An inertial tracking device was used to capture standard deviation orientations of the sensors. Accuracy of the inertial sensor

system was assessed with a goniometer before the trials began. In this sample, full thoracic range of motion T1 to T12 was recorded while standing at rest with a mean of 20.5±6.5° (Hajibozorgi and Arjmand, 2016).

In an exercise-related study by Houplin (2014), thoracic flexion was measured between high intensity interval training exercise bouts. During exercise recovery, thoracic flexion was measured via inclinometers on T1 and T12 in a standing position with the hands on the knees. The subjects performed high intensity sprinting intervals with four rest periods. Thoracic flexion increased from the first rest period at 14.6±4.4 degrees to the fourth at 19.5±8.2 degrees (Houplin, 2014).

Van Blommestein et al. (2012) used inclinometers to measure thoracic kyphosis, lumbar lordosis, and straight leg raise. Thirty healthy subjects were assessed on two occasions with a one-hour break interval. Two inclinometers were used. One was placed on T1 and T2 and the other was placed at T12 and L1. For thoracic kyphosis, interclass correlation coefficients for average measures were greater than 0.75 (good reliability). Therefore, inclinometers were a reliable measurement method (Van Blommestein et al., 2012).

Tidal Volume during Recovery. V_T is generally increased by moderate exercise. In a 2001 study by Ohashi, Kamioka, and Matsuoka, 15 healthy men, age 19-33 years, performed moderate exercise and respiratory patterns were measured during the post-exercise recovery phase. The subjects were split into four groups according to their respiratory movement patterns during recovery. Chest and abdominal expansion was recorded in the anterior-posterior direction at the sternoxiphoid process and just above the umbilicus. In accordance with the area that expanded more, the four groups included the abdomen group, partly abdomen group, abdomen-chest group, and the chest group. The subjects performed a 30-second cycle ergometer exercise

with an unspecified workload. The mean V_T in milliliters at one, two, three and four minutes post-cycling were 247, 126, 91, and 87 (abdominal group), 295, 182, 135, and 138 (partly abdominal group), 243, 128, 110, and 101 (abdomen-chest group) 254, 127, 101, and 89 (chest group), respectively. The mean HR in beats per minute at the same intervals were 150, 114, 114, and 117 (abdominal group), 173, 152, 141, and 139 (partly abdominal group), 135, 125, 128, and 117 (abdomen-chest group), 158, 130, 130, and 128 (chest group), respectively. V_T tended to be higher in the partly abdomen group in which the rate of increase in abdominal expansion was higher than the other compared groups. The authors suggest that this observation may imply that V_T during recovery is likely increased by abdominal expansion rather than thoracic expansion. Furthermore, in the abdomen group, HR was significantly lower than the partly abdomen ($p < 0.01$) and chest group ($p < 0.05$). The authors suggest that adopting certain respiratory movement strategies may benefit energy efficiency (Ohashi, Kamioka, & Matsuoka, 2001).

Tidal Volume and Sagittal Spinal Position. The direct effect of thoracic flexion on V_T was not found when searching the literature. However, a few studies on the effects of full spinal flexion on V_T were available. V_T tends to increase with spinal flexion (Paek et al., 1990). Paek et al. (1990) measured V_T during spinal flexion-extension maneuvers in five healthy male subjects. The study used respiratory inductance plethsmograph (RIP) belts to measure the cross sectional area of the rib cage and abdominal compartments. Changes in lung volume were measured with a spirometer. Lung volume was measured during the full range of spinal flexion, but divided into four equal parts at 25, 50, 75 and 100% flexion. During spinal flexion, as one thoroabdominal boundary is pushed in, the other is pushed out and the diaphragm cephalad is displaced, thus expanding the rib cage.

In a pilot study by Landers et al. (2003), breathing frequency (f_b), minute ventilation (V_E), and V_T were assessed in 17 females and 13 males. An upright sitting posture was compared to a slumped sitting posture. Contrary to the previous study, V_T and V_E were significantly increased in the upright posture. No significant difference was found in f_b. However, V_T and V_E L/min did show more of an increasing trend in the slumped sitting posture throughout the five minutes of testing. V_T and V_E increased from 0.52±0.06 L and 7.21±0.66 L/min in minute-one to 0.59±0.06 L and 7.72±0.65 in minute-five. It should be noted that eight of the subjects were considered mildly obese according to their BMI (Zerah et al., 1993). Obesity can change breathing mechanics, as well as decrease lung volume (Bray, 1985; Zerah et al., 1993). Also, this study did not measure thoracic flexion. They only instructed the subject to be fully slumped.

Lee, Chang, Coppieter, and Hodges (2010) also examined the effects of sitting posture on V_T. They had four groups of sagittal plane spinal angles. The experimenters named these reference, self-selected, slump, and thoracolumbar extension. The reference and slump postures had no group difference in thoracic angle (∠T1 T7 T12). However, the slump group had much more flexion at the thoracolumbar angle (∠T7 T12 L3) and lumbar angle (∠T12 L3 S2). The self-selected group had more thoracic extension than the slump and thoracolumbar extension groups. The thoracolumbar extension group had even more thoracic extension than the self-selected group. There were no significant differences in V_T between all four postures ($p > 0.35$).

Respiratory Mechanics and Zone of Apposition (ZOA). Compliance and lung ventilation in breathing are a result of thoracic mobility as well as excursion of the diaphragm. The movement in the thorax and the ribs allow the thorax to expand during inspiration and return to resting during exhalation (Landers et al., 2003). Different muscles are used to assist breathing during exercise when compared to resting. During rest, the diaphragm is the main breathing

muscle and contracts to assist inspiration and relaxes for expiration. During exercise, other muscles assist the diaphragm with inspiration; these include the scalenes and the intercostals (Guenette & Sheel, 2007; Roussos, 1985). Abdominal muscles assist with expiration; these include the internal oblique abdominis, external oblique abdominis, rectus abdominis, and transverse abdominis. This assistance is due to them having an advantageous point in the muscle length-tension relationship (De Troyer & Estenne, 1988; Roussos, 1985).

The abdominal muscles also control rib cage position. The position of the rib cage affects the amount of tension on the diaphragm and the ZOA (Hruska, 1997). The ZOA is a mechanical connector of the rib cage and the diaphragm. Mechanical efficiency of the muscles involved in breathing are dependent on the ZOA (Boynton et al., 1991; Hruska, 1997; Mead, 1979). The abdominal muscles are the antagonist to the diaphragm as well as rib cage expansion (De Troyer & Estenne, 1988; Hruska, 1997). The ZOA is maximized by the antagonistic action of the involved abdominal muscles (Hruska, 1997). Thoracic flexion may increase the zone of apposition (ZOA) and more thoracic extension may decrease it (Lee, 1993).

Anaerobic Power. The reliability of the Wingate test (WT) measured by the test-retest coefficient is good for peak power (P_{max}) ($r > 90$) and MP ($r > 0.91 - 0.93$) (Bar-Or, 1987; Neptune & Kautz, 2001; Patton, Murphy, & Frederick, 1985). Test-retest reliability for the WT tends to be higher for MP than P_{max} (Bar-Or, 1987). The WT conditions may also be relevant to other sport performances with intervals of high intensity exercise, like ice hockey, which has a similar fatigue curve in their skating tests (Cox, Miles, Verde, & Rhodes, 1995). The WT has been used in previous studies to examine exercise recovery (Dupont, Moalla, Matran, & Berthoin, 2007; Harbili, 2015; Lopez, Smoliga, & Zavorsky, 2014; Millar, Rakobowchuk, McCartney, & MacDonald, 2009). Repeated WTs are often used to compared recovery variables

because the test is standardized and the intensity of the recovery interval can be quantified objectively (Lopez, Smoliga, & Zavorsky, 2014).

Dupont, Moalla, Matran, and Berthoin (2007) assessed the effects of different recovery intensities on the performance of two WTs. Subjects either recovered between repeated WTs passively, at 20% maximal aerobic power, or 40% maximal aerobic power. MP and P_{max} were significantly higher after a passive recovery interval (517±26 W and 1086±153 W, respectively) when compared to active recovery intervals at 20% (484±30 W and 973±112 W, respectively) and 40% (492±35 and 928±116 W, respectively). However, subjects only had a 15-second recovery interval. Other studies with a longer, four-minute recovery time between WTs found that active recovery leads to better performance (Lopez, Smoliga, & Zavorsky, 2014; Spierer et al., 2004). However, Lopez, Smoliga, and Zavorsky found that active recovery leads to 0.6 W/kg lower P_{max} only from the first to second WT. MP output during sprint five was 6.3 W/kg in the active recovery condition and 6.0 W/kg in the passive condition. In the sixth sprint, MP was 6.5 W/kg for the active condition and 6.0 W/kg for the passive condition. Speirer et al. (2004) found that MP was significantly higher ($p<0.05$) with active recovery in sedentary subjects, but not in moderately trained hockey players. The active recovery condition had a MP of 388±42 W and the passive recovery group had a MP of 303±37 W. However, total work was higher in the active condition for both sedentary subjects and hockey players. Total work achieved in active versus passive recovery was 34890±3768 and 27260±3364 J ($p<0.02$) in the sedentary subjects and 86763±9151 and 75357±8281 J in the hockey players ($p<0.05$).

Harbili (2015) examined the effect of recovery duration between repeated Wingate tests (WT) on $P_{max,}$ MP, and the FI on elite male cyclists. P_{max} significantly decreased in repeated WTs with recovery intervals of one (-70.42 W) and two minutes (-49.73 W), but did not significantly

change with three-minute recovery intervals (-19.06 W). MP deceased in all recovery durations. For one-, two- and three-minute recovery durations, MP decreased from 590.14 to 468.01 W, 591.37 to 468.00 W, and 585.09 to 503.51 W respectively. The FI significantly decreased for one minute ($p<0.05$), but did not significantly change with two or three-minute recovery intervals ($p>0.05$). FI increased from 41.14 to 52.16 %, 45.25 to 48.56%, and 42.54 to 49.15% in the one, two- and three-minute recovery groups. The duration of recovery is a key factor in WT fatigue and P_{max} (Harbili, 2015). Type of recovery, whether passive or active, was not specified.

Cardiorespiratory Coupling (CRC). Through a phenomena called respiratory sinus arrhythmia (RSA), HRV is closely connected with external respiration (Yasuma & Hayano, 2004). CRC includes phenomena resulting from shared inputs, common rhythms, and complimentary functions (Dick et al., 2005). During gas exchange, there is a reciprocal interaction between autonomic and respiratory control systems (Dick et al., 2014). The physiological purpose of this coupling may be to increase efficiency of gas exchange by matching pulmonary perfusion to ventilation during inspiration (Hayano, Yasuma, Okada, Mukai, & Fujinami, 1996). Respiratory sinus arrhythmia (RSA) is an example of cardiorespiratory coupling (Dick et al., 2014). RSA is defined as the synchronization of HRV with respiration. The R-R time interval on an ECG is shorter during inspiration and longer during expiration (Yasuma & Hayano, 2004). RSA exemplifies the changes in HRV in relation to respiration (Prinsloo, Rauch, & Derman, 2014).

Depending on frequency, breathing affects both HF and LF variables of HRV. At about six breaths per minute (resonance frequency), changes are observed in the LF variable (Vaschillo, Vaschillo, & Lehrer, 2004). Stretch receptors are stimulated with inspiration and inhibit the medullary respiratory center and cardio-inhibitory center, thus decreasing cardiac

vagal flow (Taha et al., 1995). Also, the SA node is affected when the right atrium is mechanically stretched (Slovut et al., 1998). Both of these mechanisms result in an increased HR and RSA (Taha et al., 1995, Guzzetti et al., 1995).

Another RSA mechanism occurs at resonance frequency when inspiration decreases intrathoracic pressure (Berntson et al., 1997). The decreased pressure causes an increase in stroke volume, cardiac output, and blood pressure (Innes, De Cort, Kox, & Guz, 1993; Toska & Eriksen, 1993; Triedman & Saul, 1994). These changes result in a decrease in HR through the baroreflex loop. At resonance frequency, HR and breathing frequency are in sync, but BP is about half of a cycle out of sync (Vaschillo, Lehrer, Rishe, and Konstantinov, 2002). The time delay from the increase in BP and HR causes the HR to compensate by increasing. HR is also increasing due to the response from the stretch receptors in the atria of the heart detecting mechanical stretch. These HR responses result in maximal RSA at resonance frequency (Van Ravenswaaij-Arts, 1993).

HF is regulated by efferent vagal flow, and not affected directly by the SNS. This is because the HR response to the SNS is too slow (Fouad et al., 1984; Martinmäki et al., 2006; Pagani et al., 1997). SNS activity may have an indirect effect (Taylor et al., 2001). Another mechanism creating RSA is the cyclical vagal discharge from the medulla. This affects LF at resonance frequency and HF when breathing frequency is above nine breaths per minute (Pegani et al., 1986; Médigue et al., 2001). Also, oscillations in the respiratory and cardiovascular medullary respiratory centers affect both HF and LF in HRV (Mallani, Pagani, Lombardi, & Cerutti, 1991).

RSA may positively influence gas exchange by matching the amount of time it takes to deliver blood to the alveoli with respiration (Yasuma & Hayano, 2004). Hayano, Yasuma,

Okada, Mukai, and Fujinami (1996) demonstrated this in a study done with seven anesthetized dogs. Artificial RSA was mimicked by creating negative pressure with a diaphragm pacing technique and respiration-linked HR fluctuations using electrical stimulation of the vagi. Vagal stimulation was performed during inspiration, expiration, or constantly (control). The inspiration and expiration groups had a 4% higher increase in oxygen (O_2) uptake than the control group. The artificial RSA also decreased the ratio of physiological dead space to tidal volume by 10%. It also decreased the physiological shunt to cardiac output by 51%. This may provide evidence that RSA is beneficial for O_2 uptake by matching perfusion to respiration (Hayano, Yasuma, Okada, Mukai, & Fujinami, 1996).

HRV and Tidal Volume. Another example of CRC is the correlation of HRV with tidal volume (V_T). Pöyhönen, Syväoja, Hartikainen, Ruokonen, and Takala (2004) conducted a study on the effect of carbon dioxide (CO_2), respiratory rate, and V_T on HRV. The study included a group of 22 awake, male and female non-patient volunteers and 25 anesthetized female patient volunteers admitted for gynecologic surgery. V_T was mechanically manipulated in both anesthetized patients and non-patient volunteers. HRV was measured during spontaneous and mechanical breathing. They found that all three variables (CO_2, respiratory rate, and V_T) modulated HRV. When manipulating V_T during spontaneous breathing, volunteers were asked to increase and decrease their V_T by 20% from baseline for 10 minutes while respiratory rate and end-tidal CO_2 were constant. They found an increase in the high frequency (HF) component of HRV with greater V_T. The increase in HF was found during spontaneous breathing only and not during mechanical ventilation. With increased V_T, ln HF increased by about 3.1% and with a decrease in V_T, ln HF decreased by about 12.5%. The LF/HF ratio decreased by about 20% with increased V_T and increased by about 50% with decreased V_T, however this was not significant (*p*

> 0.01). The decrease in breathing frequency from 12 breaths•min^{-1} to 8 breaths•min^{-1} increased the LF/HF and HF in all study groups.

Other Influences on HRV. HRV is mostly modulated by the arterial baroreflex loop (Bernardi et al., 1994; Sleight et al., 1995). An increase in blood pressure (BP) stimulates baroreceptors in the aorta and carotid arteries. The impulses from these sensory receptors reach the medulla oblongata, causing a reduction in sympathetic activity therefore reducing heart rate (HR) and BP (Eckberg, Nerhed, & Wallin, 1985). The time it takes for BP to be reduced following the first increase in BP is called the Mayer wave (Bernardi et al., 1994, Julien, 2006). These waves stimulate the baroreflex causing oscillations at a low frequency (LF) (Julien, 2006; Moak et al., 2009). The decreased BP stimulates the baroreceptors, and the loop proceeds again to increase BP.

HRV is also influenced by psychological stress and fatigue (Aubert, Seps, & Beckers, 2003; Chandola, Heraclides, & Kumari 2010). Thirty healthy subjects underwent a psychological stress test that was used in a competitive setting to produce psychological strain (Delaney & Brodie, 2000). There was a significant decrease in HF after the competitive stress test. The researchers found that HRV was a suitable measure to detect the change in sympathovagal balance due to psychological stress. In another study done on fatigue, HRV was a physiological signature of fatigue in truck drivers. The HRV spectrum analysis gave a direct relationship between HRV and fatigue. LF/HF decreased with an increase in fatigue. The average LF/HF ratio was 1.8±1.15 in an alert state and 1.2±0.87 in a fatigued state. Therefore, the LF/HF ratio can be used to indicate fatigue (Patel, Lal, Kavanagh, & Rossiter, 2011). Sleep duration should also be controlled when examining HRV. Castro-Diehl et al. (2016) found that subjects who

slept less than seven hours per night had a natural log (ln) HF at baseline of about 0.31 ms^2 less than those who slept for seven or more hours.

Caffeine can also have an influence on HRV data (Bunsawat, White, Kappus, & Baynard, 2015). Eighteen healthy individuals ingested either 400 mg caffeine or placebo pills before a maximal exercise test. Those who ingested caffeine had less of an increase in ln LF/ln HF at five and 15 minutes post-exercise than the placebo group when compared with baseline values. No table was given for HRV data, only a graph, so ln LF/ln HF was roughly 2.2 ms^2 for the caffeine group and 0.12 ms^2 for the placebo group at five minutes post-exercise. Both groups had about the same ratio after 30 minutes of recovery. No significant differences were found between groups in the percent HRR ($p<0.05$). Percent HRR was calculated with the following equation: $\%HRR = (HR_{max} - HR_{1min})/HR_{max} \times 100$. %HRR in the placebo group was 14.3±1.6 bpm and in the caffeine group 14.7±1.6 bpm.

HRV in Exercise. Training produces a long-term HRV effect. A review of clinical research reported that, typically, aerobic exercise programs increase resting HRV (Prinsloo, Rauch, & Derman, 2014). More specifically, time-domain variables and HF tends to be higher in trained individuals. LF has less consistent results and may increase or decrease with training (Achten & Jeukendrup, 2003). A study done in untrained older and younger men, a six-month aerobic training program of walking, jogging, and bicycling increased resting SDNN in both older and younger men (Levy et al., 1998). This increase in resting HRV in younger men was also found in a study which compared eight trained ($VO_{2max} \geq 55$ ml/kg per min) with eight untrained ($VO_{2max} \leq 40$ ml/kg per min) men. The trained men had a higher HF, LF, and SDNN than the untrained. HF was 318±193 ms^2 for the trained men and 1,399±776 ms^2 for the untrained (Goldsmith, Bigger, Steinman, & Fleiss, 1992). Heart failure patients also show an

increase in resting HRV with aerobic training. Subjects bicycled at 60-80% of their maximum HR for 8 weeks, 5 days per week and 20 minutes per day. Average 24-hour SDNN improved from 109.3±8.3 ms pre-training to 125.6±9.0 ms post-training (Coats et al., 1992). Conflicting results showed that resistance training had no significant effect on resting HRV in healthy young adults. Twenty-two subjects performed an eight-week high-intensity whole-body strength training protocol. However, SDNN did somewhat increase from 59±9.5 to 63±9.8 ms pre- to post-training (Cooke & Carter, 2005).

During exercise, sympathetic tone increases (Ekblom, Kilbom, & Soltysiak, 1973) and parasympathetic tone decreases (Pickering, Gribbin, Peterson, Cunningham, & Sleight, 1972). Ekblom, Kilbom, and Soltysiak (1973) conducted a study where they gave propranolol, a beta blocker, to subjects before exercise. The decrease in HR was less pronounced when exercising versus at rest, so it may be assumed that exercise increases sympathetic tone (Ekblom, Kilbom, & Soltysiak, 1973). Pickering, Gribbin, Peterson, Cunningham, and Sleight (1972) studied the effects of propranalol during exercise and at rest. They found that baroreflex regulation of pulse interval may be modulated by sympathovegal balance acting on the sinoatrial node, since atropine blocked the baroreflex response during exercise, and propranolol had no effect (Pickering, Gribbin, Peterson, Cunningham, & Sleight, 1972). This may show that the reflex is mediated by parasympathetic nerves. The sympathetic response (an increased HR) declines with increased parasympathetic tone. Some parasympathetic tone persists up to a HR of 190 bpm, so even during exercise, some parasympathetic response is still in effect (Pickering, Gribbin, Peterson, Cunningham, & Sleight, 1972). It is best to start HRV measurement immediately after exercise to capture the rapid increase at the beginning of recovery due to the rise in

parasympathetic activity (O'Leary, 1993). The increase in HRV then slows down later as sympathetic withdrawal occurs (Kluess, Wood, & Welsch, 2000).

Javorka, Zila, Balhárak, and Javorka (2002) conducted a study to examine the relationship of HRV with the reduction in heart rate (HR) after exercise. They collected heart rate activity with an ECG. Seventeen healthy males laid supine for 25 minutes, stood for 5 minutes, performed a step test at 70% W_{max} for 8 minutes, and lastly they rested in supine to recover for 35 minutes. Javorka at al. found that SDNN, HF, and LF continuously increased during the recovery phase immediately post-exercise. SDNN, HF, and LF all remained lower than pre-exercise levels for at least 30 minutes compared to the first supine rest phase. They found no difference in LF upon standing. The experimenters also found that HRR was positively correlated with HRV at 5 and 10 minutes from the onset of recovery. All HRV components increased more rapidly at the beginning of recovery from about 330 s post-exercise and started to slow down later at about 1200 s. SDNN increases as parasympathetic tone increases back to normal with recovery (Javorka, Zila, Balhárak, & Javorka, 2002).

HRV during Recovery. HRV during exercise recovery is affected by training status and body position. In sedentary subjects, LF/HF during supine recovery took longer than an hour to reach baseline after supramaximal exercise (Stuckey et al., 2012). However, when the exercise session is low intensity and short duration, pre-exercise HF and RMSSD can be reached within 5 minutes in highly conditioned athletes. Highly trained male runners were able to return to resting HF and RMSSD 5-10 minutes after running 60 or 120 minutes at an intensity below ventilatory threshold (Seiler, Haugen, & Kuffel, 2007).

Guerra et al. (2014) had a sedentary group, an aerobically trained group, and a resistance-trained group. The aerobically trained were the only subjects who experienced vagal reactivation

within four minutes after a maximal progressive cycling test. The test started at 25 W and increased by 25 W every minute maintaining 50-60 revolutions per minute cadence. The test was terminated when three of the following criteria were met: failure to maintain the selected cadence, RPE > 18 on the Borg's 6-20 scale, respiratory exchange ratio > 1.1, and HR > age 95% of age-predicted maximum (220-age). RMSSD was measured every 30 seconds post-exercise for 300 seconds and vagal reactivation was defined as statistical difference from the first 30 second interval post-exercise. This occurred at 210 seconds on average. Both resistance and aerobically trained groups experienced faster heart rate recovery (HRR) than the sedentary group. Only the aerobically trained group showed a significant increase in RMSSD during recovery. A significant change occurred 210 seconds relative to the 30 seconds post-exercise value ($p < 0.05$). Therefore, it appears that recovery of the RMSSD variable of HRV may be a measure of aerobic fitness.

Body position during recovery and HRV has been examined by Barak et al. (2010) on healthy, untrained young men. These men were assigned to three different recovery position groups: supine, supine with elevated legs, and seated (Barak et al., 2010). The subjects performed five minutes of cycling on an ergometer at 80% of the peak HR achieved during a previous Wingate Test. Restoration of pre-exercise HRV was not present within 15 minutes after exercise, regardless of body position (Barak et al., 2010). Time domain HRV variables, RRNN and RMSSD, were higher in supine and supine with elevated legs positions during recovery. Pre-exercise, the natural logarithm of HF (ln HF) was significantly higher in supine by 12.5% ($p < 0.01$) and supine with elevated legs by 7.2% compared to the seated position. The seated position had the lowest ln HF of the three positions. During recovery, there was no significant difference in the post-exercise ln HF between the three different body positions ($p > 0.05$).

Summary

From this review, the interplay between the autonomic nervous and respiratory systems are reviewed. The possible effects related to thoracic flexion on these two systems are also presented. The ability of the autonomic nervous system to better adapt to the cardiovascular and respiratory stresses of exercise can be examined by measures of parasympathetic reactivation, HRR and HRV. Earlier parasympathetic reactivation may be a predictor of subsequent athletic performance (Daane, Lamberts, Kallen, Jin, & Van Meeteren, 2012; Guerra et al., 2014; Lamberts et al., 2010; Stuckey et al., 2012).

Athletic performance may also be predicted by modulations in the respiratory system; therefore respiratory variables may be important factors to examine during recovery (Amann, 2012). Increases in the HF variable of HRV and thoracic flexion are both correlated with an increase in V_T (Paek, Kelly, & McCool, 1990; Pöyhönen, Syväoja, Hartikainen, Ruokonen, & Takala, 2004). Currently, there are no studies that directly measure the effect of thoracic flexion on HRR. The current study aims to determine if recovery, quantified by HRR, VCO_2, and V_T can be altered by thoracic flexion. Another aim is to see whether or not these recovery variables can predict performance by comparing the change in MP and FI after recovering from exercise with thoracic flexion at 14 degrees or greater versus a neutral thoracic spine.

Chapter III

Methods and Procedures

Introduction

The purpose of this study was to determine if thoracic position during cycling sprint recovery has an influence on recovery variables, such as heart rate recovery (HRR), carbon dioxide production (VCO_2), and tidal volume (V_T). This study also examined the influence of thoracic position on subsequent performance, measured as the change in mean anaerobic power (**Δ**MP) and fatigue index (**Δ**FI) from the first (WT_1) to the second (WT_2) Wingate anaerobic test. Specifically, this study compared the results of two spinal position conditions: a neutral thoracic condition (NC) verses a flexed thoracic condition of ≥ 14° (FC) during exercise recovery (Houplin, 2014).

This chapter describes the methods and procedures used for this study. Included are a description of the subjects, the design of the study, and data collection procedures. The data collection section specifies instrumentation, measurement techniques, procedures, and statistical analysis.

Description of study population

The subjects included 13 males, age 21-44 years. All were apparently healthy competitive cyclists who had completed at least one cycling race within the previous 12 months. Cyclists were chosen because they are familiar with cycling exercise. Excluding criteria were

hypertension, smoking, long-term inhaler use, diabetes, high blood pressure, obesity, cardiovascular disease, and pulmonary restrictions. Participants were recruited by flyers posted in the local bicycle retailers. Exclusion criteria was detailed and distributed via e-mail to the participants at the time of recruitment. Before testing, all subjects were provided with an informed consent document and made aware of the testing procedures (Appendix A). The university's Human Subjects Committee reviewed and approved the study prior to any data collection (Appendix A).

Design of study

This study utilized a pretest-posttest experimental design in which subjects were randomly assigned on the first session to assume one of two conditions. The conditions randomized were either NC or FC during the recovery intervals. Target thoracic flexion for FC and NC was taken on the bike. For the FC, participants were instructed to maintain the target maximal thoracic flexion and for NC their minimal flexion. In the present study, FC thoracic flexion from T1 to T12 was considered to be an angle of 14° or more. The subjects started with a five-minute warm-up at 75 W, followed by WT_1, a four-minute active recovery interval pedaling at 75 W in the assigned posture, WT_2, and then another four-minute active recovery interval at 75 W. Participants self-selected the cadence for the first warm-up and this same cadence was held consistent for the following recovery intervals as well as the second testing day. After no less than a 48-hour period, subjects participated in the second session where they followed the same procedure, but performed the other condition. MP and FI were measured during WT_1 and WT_2. HRR was measured during the first minute of both recovery intervals. VCO_2, and V_T were measured every minute at 30, 60, 150, and 210 s during both recovery intervals and the average of the four measurements were used for analysis.

Data collection procedures

Instrumentation. HR was recorded with a Polar heart rate monitor (Lake Success, NY). VCO_2 and V_T were measured with a Parvomedics TrueOne Metabolic Cart (Sandy, UT). The Wingate Anaerobic Test was performed on a Velotron cycle ergometer (Racer-Mate Inc., Seatlle, WA) for measurement of MP and FI. A goniometer with a built-in leveler was used to measure trunk angle during participant set-up on the cycle ergometer, Thoracic flexion was measured every 30 seconds with spine inclinometers at T1 and T12 to assure real-time consistency of flexed and neutral positions (Van Blommestein, MaCrae, Lewis, & Morrissey, 2012).

Measurement techniques and procedures.

Thoracic position was randomized by the flip of a coin for the first day of data collection. Subjects were either instructed to maintain a NC or FC during the exercise recovery portions. The NC was held consistent and confirmed by inclinometers placed at T1 and T12. Target thoracic flexion for NC and FC on the bike was taken on the first data collection day before testing. To get the target degrees on thoracic flexion for FC, participants were asked to arch the spine up between the shoulder blades as much as possible, while still being comfortable. For NC, participants were instructed to flatten their spine between the shoulder blades as much as possible, while still being comfortable. Before both target thoracic flexion measurements, participants were instructed not to change hip angle, but keep the movement in the upper back only. During the recovery intervals, the experimenter measuring thoracic flexion with inclinometers gave real-time feedback to the participant every 30 seconds whether they were under or over their baseline maximal flexed thoracic position for FC or their baseline minimal flexed thoracic position for NC. When subjects tested in the FC, they needed to hold thoracic flexion at 14° or greater.

The ergometer set-up was standardized to a cycling position for internal validity and consistency. The distance from the top of the saddle to the pedal surface or seat height was set to 100% of trochantric length (Nordeen-Snyder, 1977). The handlebar height was adjusted to the same level as the seat height. Handlebar fore-aft position was modified to achieve a trunk angle of 20 to 30° (Ericson, Bratt, Nisell, Arborelius, & Ekholm, 1986; Korff, Newstead, Zandwijk, & Jensen, 2014). The cycle ergometer set-up was recorded for each participant and adjusted to the same position for the second testing day.

Subjects were instructed to stay seated and keep their hands in the tops position during the whole protocol. Subjects completed a five-minute warm-up at a self-selected cadence at 75 W. Subjects then completed WT_1 followed by a four-minute active recovery interval at 75 W and the same cadence in the thoracic posture selected for that session. Then, they performed WT_2 at their usual or comfortable self-selected thoracic flexion. For WT_1 and WT_2, the subjects were instructed to pedal as fast as they could for a 30 second period at 0.075 kg per kg of body mass and were given verbal encouragement. The cadence selected was recorded and the subject was asked to keep this consistent throughout both recoveries. Cadence was displayed on a screen that the subject could read.

Each subject once again assumed either the FC or NC spinal position and pedaled at the same cadence selected during the warm-up at 75 W. HRR, VCO_2, and V_T were all recorded during the four-minute recovery data collection. HRR was recorded for the first minute of recovery following both WT_1 and WT_2, as in similar research (Lamberts, Lemmink, Durandt, & Lambert, 2004; Lamberts, Swart, Noakes, & Lambert, 2008). VCO_2 and V_T were recorded at one-minute intervals during the passive recovery interval after WT_2. The metabolic cart was used simultaneously to measure VCO_2 and tidal volume via a two-way breathing mouthpiece. These

measures were recorded every minute during the recovery period and the average was used for analysis

Data processing. V_T and VCO_2 were obtained through the metabolic cart at one-minute intervals for four minutes at 30, 90, 150, and 210 s following WT_1 and WT_2. The average V_T and VCO_2 for each four-minute recovery period were used for analysis. HRR was calculated as the difference in HR from the end of each WT to one minute later. The MP, FI, HRR, V_T, and VCO_2 data were transferred into Excel (Microsoft Inc., Redmond, WA) for data analysis. FI was calculated by the Racermate software as [(max power – min power)/test duration] and shown in W/s. ΔMP and ΔFI from WT_1 to WT_2 were calculated in Excel. Subjects unable to achieve greater than 14° thoracic flexion in the FC were to have their data excluded from data processing.

Data Analysis

Dependent t-tests were performed to compare ΔFI, ΔMP, HRR, V_T, and VCO_2 in neutral verses flexed thoracic recovery positions. The average degree of thoracic flexion was used for FC and NC recovery intervals. Statistical significance was established at $p < 0.05$. Effect size was calculated. Cohen's d was used to indicate the standardized difference between the two means. Data analysis was performed on Excel (Microsoft Inc., Redmond, WA) and IBM SPSS 23 (Armonk, NY).

Chapter IV

Results and Discussion

Introduction

The purpose of this study was to investigate the effects of thoracic spinal position on recovery between cycling sprints. The design of the study was a pretest-posttest experimental design. On separate days, participants recovered from two cycling sprints (WT_1 and WT_2) in one of two conditions. They recovered in either a neutral thoracic spinal position or a more flexed thoracic spinal position. The two conditions were referred to as the neutral condition (NC) and the flexed condition (FC). The dependent variables included tidal volume (V_T), carbon dioxide production (VCO_2), heart rate recovery (HRR), change in mean power (ΔMP), and change in fatigue index (ΔFI). This section presents the results of the subject demographics, dependent variables, and the amount of thoracic flexion in each condition.

Subject Demographics

Thirteen male competitive cyclists, between the ages of 21 and 44, participated in this study. Competitive cyclists were chosen because they are familiar with the cycling exercise. Subjects took part in anywhere from 1 to 26 competitions in the twelve months prior to participating. Subjects acted as their own control. They were asked to avoid unusual exercise that may create delayed onset muscle soreness for testing and get at least seven hours of sleep the nights prior to both testing days. All of the 13 participants completed both testing conditions.

Table 1 details subject demographics including age, weight, height, and the number of competitions completed. Also detailed in Table 1 are the target degrees of TF for each condition.

Target TF for each condition were measured on the bike before collection on the first day of testing. Subjects assumed their most flexed for FC and most extended for NC thoracic spinal postures while still remaining comfortable. These target TF measurements were used as a guideline for one experimenter to give real-time feedback during recovery keep the participant in a consistent posture.

Table 1. Participant demographics (mean ± SD) including age, weight, height, the number of cycling competitions completed in the 12 months prior to data collection, and their target FC and NC degrees of TF.

Age (years)	Weight (kg)	Height (m)	Cycling Competitions	Target FC TF (deg)	Target NC TF (deg)
30.8 ± 7.1	75.5 ± 6.6	1.8 ± 0.1	9.5 ± 7.4	49.9 ± 5.1	30.0 ± 9.1

Results of the Study

There were no significant differences (p>0.05) in HRR, VCO_2, V_T, ΔFI or ΔMP between conditions. There was, however, a significant difference (p<0.001) between conditions in the degrees of thoracic flexion (TF) held during recovery. Table 2 shows the average values of HRR, VCO_2, and V_T, during both of the active recovery phases. Table 3 shows the average ΔFI and ΔMP from the first (WT_1) to the second (WT_2) Wingate Anaerobic Test. Cohen's d was calculated from the mean and standard deviations listed in Table 2. As expected, there was a large effect of thoracic flexion (p=0.000; d=1.71). A small effect was found on HRR (p=0.293; d=0.33), V_T (p=0.121; d=0.36), and ΔFI (p=0.289; d=0.44). All statistics are detailed in Appendix G.

Table 2. TF, HRR, V_T, and VCO_2 (mean ± SD) during both recovery intervals for FC and NC

	Flexed Condition	**Neutral Condition**	***p*-value**	**Cohen's d**
TF (deg)	45.6 ± 7.3	32.1 ± 8.4	0.000*	1.71◆◆
HRR (bpm)	23.5 ± 7.8	21.3 ± 5.0	0.293	0.33◆
V_T (L)	3.00 ± 0.51	3.19 ± 0.55	0.121	0.36◆
VCO_2 (L/min)	3.28 ± 0.26	3.26 ± 0.40	0.794	0.06

*indicates significance $p<0.05$; ◆◆indicates a large effect and ◆indicates a small effect between means using Cohen's d.

Table 3. ΔMP and ΔFI from WT_1 to WT_2 (mean ± SD) for FC and NC

	Flexed Condition	**Neutral Condition**	***p*-value**	**Cohen's d**
ΔMP (W)	-29.7 ± 17.5	-28.8 ± 18.9	0.853	0.051
ΔFI (W/s)	0.59 ± 3.61	-0.43 ± 1.94	0.289	0.44◆

◆indicates a small effect between means using Cohen's d.

Discussion

The purpose of this study was to explore the effect of thoracic spinal position on cycle sprint recovery and subsequent performance. No significant differences were found between conditions in any of the recovery or performance variables. However, there were some small effects of condition on HRR, V_T, and ΔFI. There is no other research on the effect of thoracic spinal position on cycle sprint recovery. The current results could not be compared directly to any other research, however there are some studies that examined the effects of spinal position

on HRR, V_T, and VCO_2 (Houplin, 2014; Paek et al., 1990; Slutsky et al., 1981). Other studies have compared these recovery variables with differences in performance.

In the present study, average thoracic flexion in FC was 45.6 ± 7.32 deg and in NC was 32.1 ± 8.37 deg. This is quite large compared to other values obtained in the literature. For example, in a study by Houplin (2014), thoracic flexion was measured between high intensity interval training exercise bouts. Thoracic flexion was measured in the standing position and the range increased from 14.6±4.4 deg to 19.5±8.2 deg. Thus, any data less than 14 deg was chosen as an eliminating FC criterion. The present TF data was higher, possibly because the participants in the Houplin (2014) study were not actively trying to come into TF. They were just assuming a hands-on-knees posture.

Another reason TF data was higher could be that the present participants were competitive cyclists. Studies have even found that chronic spinal adaptations occur in master and elite cyclists. Thoracic kyphosis is developed in the standing position (Muyor, López-Miñarro, & Alacid, 2011; Rajabi et al., 2000). Muyor et al. (2011) found that the average elite cyclist had a standing thoracic curvature of 48.17±8.05 deg and the average master cyclist had a thoracic curvature of 47.02±9.24 deg. Target FC and NC TF data was collected because the cyclist population has more kyphosis than the average population. The present TF data was in-between non-cyclist and master cyclist data. This is fitting considering that they were in-between those groups in their training experience. However, the range of motion appeared to be similar to other literature. The difference in the FC and NC target TF for the current data had a mean of 19.9±6.8 deg. This is similar to previously recorded full thoracic range of motion with a mean of 20.5±6.5 deg (Hajibozorgi & Arjmand, 2016).

In a cyclist population with a similar mean age (30.36±5.98 years) to the present study, thoracic angle was measured on the bicycle (Muyor, Alacid, & López-Miñarro, 2011). The study was divided into three groups determined by hamstring extensibility tested by a passive straight leg raise. Those that scored <80, 80-90, or >90 deg had a mean TF of 41.03±9.69, 38.80±10.09, or 39.65±10.00 deg. There was no significance between groups (p=0.664). To compare these measurements to the present data, the midpoint between the two condition's average TF data collected in the present study was calculated as 38.9±7.8 deg. This was very similar to what was found by Muyor, Alacid, and López-Miñarro (2011).

In this study, mean HRR was 23.5±7.81 bpm in the FC and 21.3±4.98 bpm in the NC. The average difference between conditions was not significant (p>0.05). However, there was a small effect of condition on HRR according to Cohen's d (d=0.33) was found. A thesis study by Houplin (2014) also found an increase in HRR with more TF. In this study, participants recovered between high intensity interval training sprints on a treadmill in either a hands-on-head position or a hands-on-knees position. TF was also measured with inclinometers and mean TF during the hands-on-knees condition increased from 14.6 to 19.5 degrees from the first to the fourth rest interval. Mean HRR was higher in the hands-on-knees condition (53±10.9 bpm) when compared to the hands-on-head condition (31±11.3 bpm).

HRR may have been lower than other reported values because of the subject age difference. In the present study, participant age ranged from 21 to 44 years with an average of 30.8 ± 7.09 years. The study by Houplin (2014) had an average participant age of 20.3±1.1 years with a range of 18 to 22 years. HRR decreases with age in cardiac patients (Sydó et al., 2015). However, in other studies on healthy populations, only training status, not age, was found to affect HRR (Borges et al., 2017; Darr et al., 1988). Another reason HRR may have been lower in

the present study is that the training type differed. Most of the participants were road cyclists and trained for longer duration, lower intensity bike rides. The subjects in the Houplin (2014) study were all familiar with high intensity interval training (HIIT). In a study by Lamberts, Swart, Noakes, and Lambert (2008), already well-trained cyclists still improved their HRR (+7±6 beats; p=0.001) after doing four weeks of HIIT. Therefore, HRR may have been attenuated in the present study because most of the cyclists trained continuously at a lower intensity.

The present study found no significant difference (p<0.05) in V_T between FC (3.00±0.514 L) and NC (3.19±0.545 L). There was a small effect of condition on V_T (d = 0.36). However, Landers et al. (2003) found significantly higher V_T and minute ventilation in an upright seated posture when compared to a slumped sitting posture. The authors concluded that sitting in a slumped posture may lead to decreased oxygen delivery to the physiological systems of the body. Contrary to the results found by Landers et al. (2003), a study by Paek, Kelly, and McCool (1990) found an increase in V_T with spinal flexion. However, the participants in the study were standing during data collection. This may have changed breathing mechanics.

In previous studies, thoracic flexion has been shown to increase the zone of apposition (ZOA)(Lee, 1993). Compliance and lung ventilation in breathing are a result of thoracic mobility as well as excursion of the diaphragm. Movement in the thorax and the ribs allow the thorax to expand during inspiration (Landers et al., 2003). Therefore, it was expected to see greater V_T in FC. However, the ZOA is maximized by the antagonistic action of the involved abdominal muscles (Hruska, 1997). It could be possible that the participants had too much thoracic flexion, shortening the abdominal muscles. The shortening may reduce tension and subsequently reduce force of the abdominal muscles according to the muscle force-tension relationship. This shortening of the abdominal muscles could have effected breathing mechanics.

It was hypothesized that VCO_2 would be higher in FC in the present study. Mean VCO_2 was 3.28±0.259 L/min in FC and 3.26±0.397 L/min in NC. This was not significantly different between conditions (p>0.05) and there was no effect using Cohen's d (d=0.062). In a animal model study by Slutsky et al. (1981), VCO_2 increased with breathing frequency, even as V_T was mechanically stabilized. They found that increasing lung volume had no significant effect on VCO_2. However, when V_T was doubled in the dogs, there were varied effects on VCO_2. In some, VCO_2 decreased by 13% and in some VCO_2 increased by 110% (mean=+35%).

In the present study, breathing frequency was not controlled, however, if breathing frequency was similar between conditions, VCO_2 may be dependent on V_T. V_T was not significantly different. This may be why there was no significant difference in VCO_2. Increased VCO_2 may be a determinate of increased buffering capacity within the muscle allowing for a better glycolytic enzyme activity (Parkhouse & McKenzie, 1984; Sharp et al., 1986). The lack of difference in VCO_2 between conditions may be an absence of change in glycolysis and glycogenolysis between conditions. It may also mean that similar amounts of CO_2 were retained in the lungs for both conditions (Østergaard et al., 2012).

It was also expected that increased buffering in FC would in turn improve performance. Performance did not appear to improve as demonstrated by ΔMP and ΔFI. MP decreased by about the same amount in both condition. FI actually improved from WT_1 to WT_2 in the NC (ΔFI = 0.592±3.61 W/s) and worsened in the FC (-0.685±1.94 W/s). However, this difference was not significant. There was a small effect condition on ΔFI (d=0.44). The increase in FI in NC may be due to small sample size, the familiarity affect, subject motivation, or post-activation potentiation (PAP). A limitation to this study included the assumption that participants would try equally hard during each WT. This was instructed, however this may not have been the case for some, as

some had positive ΔFI. Another explanation for the positive ΔFI may have been PAP. It has been shown that applying a high load on the muscle could increase output by that muscle minutes later. Studies have found performance improvements with PAP followed by four minutes of rest (Duncan, Thurgood, & Oxford; Kinet, 2017). The present protocol involved a load increase on the muscle during WT_1, then a four-minute recovery interval, followed by WT_2. The load from WT_1 may have caused some participants to potentiate, thus improving WT_2. Performance increases in MP have been found with improvements in HRR (Lamberts et al., 2010). The experimenters found cyclists with improved HRR also significantly improved MP in their 40-km time trial at 20 W. There were also improvements in their 40-km time trial, however these were not significant.

Performance differences may have not been found because the present study did not control for training type. Some participants focused mostly on long duration road racing are not used to pushing themselves into a maximal effort sprint and may have done better the second testing day. Other participants were involved with cyclocross and trained at a higher intensity for shorter durations. In a study by Cheilleachair, Harrison, and Giles (2016), training type was found to affect performance and aerobic capacity. Eight weeks of HIIT was compared to long slow distance training in 19 well-trained rowers. There were greater improvements in 2000 m time trail (ES=0.25), VO_{2max} (ES=0.95; p=0.035), and power output at lactate threshold (ES=1.15, p=0.008) with HIIT.

Summary

Results of the present study indicate that thoracic spinal position during active recovery may have little to no effect on HRR, VCO_2, and V_T in competitive cyclists. No significant differences were found in HRR, VCO_2 or V_T between NC and FC. Recovery thoracic spinal

position did not make a difference ($p<0.05$) in subsequent performance, represented as ΔMP and ΔFI, from one cycling sprint to another. These results suggest that competitive cyclists who want to improve their recovery and performance during a race may not receive a benefit from using a more flexed thoracic spine position. It may be better for cyclists to focus on other methods for enhancing active recovery.

Chapter V

Summary and Conclusions

Summary

There is a lot of research on how to recover the body after a cycling performance. Many methods have been examined, including compression garments, electrical stimulation and humidity therapy (Argus, Driller, Ebert, Martin, & Halson, 2013). However, there is a paucity of research on how to recover during a race or practice immediately between cycling sprints. The only studies focused on posture during exercise recovery in healthy adults included measuring responses to standing, seated, or supine positions (Takahashi et al., 2000).

The subjects of this study included 13 competitive male cyclists recruited through local bicycle shops. Competitive cyclists were chosen because they are familiar with the cycling exercise. Limitations may have included the familiarity effect. Some cyclists who focus mostly on long duration road racing are not used to pushing themselves into a maximal effort sprint and may have done better the second testing day. Also, one condition may have been more comfortable than another. This may have affected performance due to fatigue from trying to get into a position they are not used to. Also, a there may have been a lack of motivation or difference in how the participant was feeling on one of the testing days.

In the current study, there was a small effect of thoracic spine position during recovery on heart rate recovery (HRR) and tidal volume (V_T). There was also a small effect of thoracic spine position on the change in fatigue index (ΔFI) from one sprint to another. However, there was no effect of thoracic position on the carbon dioxide output (VCO_2) or the change in mean power

output (ΔMP) from sprint to sprint. There may be little to no benefit in assuming a more flexed thoracic position between cycling sprints.

Conclusions

Based on the findings of this study, there were no significant differences in HRR, V_T, or VCO_2 between the NC and FC during cycle sprint recovery. There may have been a small beneficial effect of the FC on HRR and this may have carried over to improving ΔFI from WT_1 to WT_2. However, there was a small detrimental effect of the FC on V_T. There were no significant differences between condition or effect on ΔMP. Therefore, more flexion in the thoracic spine may have little to no benefit on cycle sprint recovery and subsequent performance.

Recommendations

Future research may want to focus on an array of positions based on the subject's thoracic spine range of motion during cycle sprint recovery. Perhaps there is an ideal or maximizing recovery position for improving performance. Future studies may also want to focus more specifically on cyclists that are used to doing maximal sprints, like cyclocross athletes. A larger amount of participants may also help researchers to identify trends more clearly. Also, a smaller participant age-range may help to identify a difference in HRR.

The ability to recover quickly from cycle sprints may serve to improve performance in a race. However, the present data shows little to no effect of the FC on cyclist recovery and subsequent performance. Some participants reported feeling uncomfortable in one condition verses the other. There is no clear advantage to either position condition. Perhaps, if there is not much of an effect, cycling athletes should choose a posture that is comfortable or aerodynamic to benefit performance.

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Appendix A

Informed Consent

CONSENT FORM

Effects of Thoracic Spine Position during Cycle Sprint Recovery

Deanna Emnott

Purpose and Benefit:

The researchers have been interested in the effects of spinal posture on cycling recovery and performance. The purpose of this study is to examine how recovery posture affects physical performance. The results of this study will advance understanding of the role of posture on cycling sprint power, breathing, and heart rate variables. This may lead to the development of improved recovery strategies for cyclists.

I UNDERSTAND THAT:

With your consent, your participation will last approximately 20 minutes per day for two days with no less than 48 hours between sessions. On the first day, you will be randomly assigned one of two spinal postures to hold during the light cycling intervals. On the second day, you will do the same series of tasks; however, you will assume the other posture (not previously assigned). The two postures include a neutral spinal posture or a slouched position. Each session will involve completion of a series of tasks that include a light cycling warm-up for 5 minutes, a 30-second maximal sprint cycling test, four minutes of light cycling, another 30-second maximal sprint cycling test, and then five minutes of light cycling. During the last five-minute bout of light cycling, a mouthpiece will be inserted and the attached headgear will be placed. During the whole series of tasks you will be wearing a heart rate monitor attached to an elastic band that fits around the rib cage.

The risks associated with participation may include discomfort due to maximal exercise tasks and breathing into a mouthpiece for breathing analysis. You may also experience light-headedness, nausea, chest pain, or vomiting. Please alert the experimenter immediately if you experience any of these symptoms. There will be at least one experimenter who is CPR, first aid and AED certified present during all data collection.

All information is confidential. My signed consent form will be kept in a locked cabinet. All data will be kept on a computer locked by a pass code. Real names will not be included in the data. Data will be analyzed by subject number and only primary researchers will have access to the records.

This experiment is conducted by Deanna Emnott. Any questions you have about the experiment or your participation may be directed to her at emnottd@wwu.edu or (425) 327-9170. You may also contact Dr. Lorrie Brilla at Lorrie.Brilla@wwu.edu or 360-650-3056.

If you have any questions about your participation or your rights as a research participant, you can contact the WWU Human Protections Administrator (HPA), (360) 650-3220. If during or after participation in this study you suffer from any adverse effects as a result of participation, please notify the researcher directing the study or the WWU Human Protections Administrator.

Your signature indicates that you have read and understand the information provided on this form. Your signature also indicates that you willingly agree to participate. Your signature on this form does not waive your legal rights for protection. Your participation is voluntary. You may choose to withdraw from participation at any time without penalty. Thank you for your participation in this study.

*I am at least 18 years of age

_____ _____

Participant's Signature Date

_____

Participant's Printed Name

NOTE: Please sign both copies of this form and retain the copy marked "Participant Copy"

Researcher Copy Participant Copy

Appendix B

Human Subjects Activity Review Form

Human Subjects Activity Review

1. **What is your research question, or the specific hypothesis?**

The current study will be conducted to determine the effect of thoracic flexion on short term maximal cycling exercise recovery and possible impacts on subsequent performance. The null hypothesis states that there is no effect of thoracic flexion on heart rate recovery (HRR), carbon dioxide production (VCO_2), and tidal volume (V_T) during maximal cycling exercise recovery. The null hypothesis also states that there is no effect of thoracic flexion in recovery on mean power (MP) or fatigue index (FI) during maximal cycling exercise.

2. **What are the potential benefits of the proposed research to the field?**

Athletic performance may be improved by enhancing recovery from training and competition. Utilizing appropriate strategies for recovery is believed to enhance performance and minimize injury risk (Fridén, Sjöström. & Ekblom, 1981; Mujika, 2012; Otter, Brink, van der Does, & Lemmink, 2016). The sport of cycling involves maximal effort sprints with active recovery intervals in-between. The respiratory and autonomic systems work hard to recover the body towards homeostasis between sprints to prepare for the next effort. Thus, improving respiration and autonomic profile during recovery may help improve performance. The direct effect of thoracic flexion on recovery has not yet been studied.

Thoracic flexion increases the zone of apposition (ZOA) and more thoracic extension decreases it (Lee, 1993). The ZOA connects the diaphragm to the ribcage mechanically (Boynton, Barnas, Dadmun, & Fredberg, 1991; Mead, 1979). An optimal ZOA may maximize the contraction of the diaphragm (Lando et al., 1999). V_T is a variable used to measure the depth

of one breath (Pöyhönen, Syväoja, Hartikainen, Ruokonen, & Takala, 2004). V_T may also be increased with thoracic flexion. A higher V_T could be beneficial to cyclists during a race.

V_T may also affect carbon dioxide expelled (VCO_2). In a study on 13 dogs, a doubling of V_T resulted in an average increase in VCO_2 of about 35% (Slutsky et al., 1981). VCO_2 is also altered by training status and aerobic fitness. Multiple sprint-type sports games players have a higher VCO_2 than endurance-trained runners during post-exercise recovery (Hamilton, Nevill, Brooks, & Williams, 1991). This may be due to greater increases in buffering capacity within the muscle, allowing for a higher potential for glycolytic enzyme activity (Parkhouse & McKenzie, 1984; Sharp et al., 1986). This may explain why multiple-sprint-type sports players also showed higher mean anaerobic power output (MP) than endurance-trained athletes (719 and 657 Watts, respectively) (Hamilton, Nevill, Brooks, & Williams, 1991).

During exercise, cardiac output increases as a result of cardiac pumping autoregulation in response of the SNS to a higher metabolic demand. The PNS then reduces cardiac output following exercise (Javorka, Zila, Balhárak & Javorka, 2002). HRR is the rate at which heart rate (HR) declines and is usually measured within minutes following a bout of exercise (Borresen & Lambert, 2007; Lamberts et al., 2008; Shetler et al., 2001). HRR improves with training (Daanen et al., 2012). The ANS regulates both the increase in HR during exercise and the decrease in HR within minutes of the cessation of physical exercise. HRR is characterized by the reactivation of the PNS and the withdrawal of the SNS (Borresen & Lamberts, 2007; Pierpont & Voth, 2004; Savin, Davidson, & Haskell, 1982; Shetler et al., 2001).

HRR improves with training, even in already well-trained cyclists. Lamberts, Swart, Noakes and Lambert (2008) put trained cyclists with an average VO_{2max} of about 60 ml $kg^{-1}min^{-1}$ and peak power output of 5.2 W kg^{-1} through a high-intensity training (HIT) program. Peak

power output in a 40-km time trial improved by 4.7%. HRR average was initially 29±6 beats and 35±4 beats post-training. Significant changes were not found in VO_{2max}. The experimenters found that HRR after the 40-km time trial correlated well with improvements in performance variables such as peak power output ($r = 0.73$; $p < 0.0001$) and 40-km time trial ($r = 0.96$; $p < 0.0001$).

Knowing whether or not thoracic flexion changes recovery variables and impacts subsequent performance may give insight on how to recover for optimal autonomic adaptation and respiratory function. Heart rate recovery will be used to measure recovery of the autonomic nervous system. Carbon dioxide expelled and tidal volume will be used to measure recovery of the respiratory system. The effect on performance will be measured by the mean power and rate of fatigue during maximal cycling tasks.

3. What are the potential benefits, if any, of the proposed research to the subjects?

This research may provide support for the use of thoracic flexion during cycling sprint recovery and the subjects may use this knowledge in future competition and training. They will also receive their peak power and mean power results to have knowledge of their fitness level to compare future performance changes.

4. A. Describe how you will identify the subject population, and how you will contact key individuals who will allow you access to that subject population or database.

The researchers will recruit subjects from the WWU cycling team and local cycling shops. With permission from the store staff, posters and flyers will be distributed at Earl's Bike Shop,

The Kona Bike Shop, The Hub Community Bike Shop, Kulshan Cycles, Fairhaven Bicycle, Jack's Bicycle Center, DNZ Performance Bike, REI, Transition Bikes, Alleycat Bike Shop, and Fanatik Bike Company. The primary researcher will be contacting the WWU cycling team captain through e-mail to relay the request to the team. The bike shop staff will be asked if posters are allowed for this research, if not, the owners will be contacted by phone or e-mail.

B. Describe how you will recruit a sample from your subject population, including possible use of compensation, and the number of subjects to be recruited.

At least 20 subjects will be recruited for this study. Subjects must be current competitive cyclists who have competed within the last year. Excluding criteria are hypertension, smoking, long-term inhaler use, diabetes, high blood pressure, obesity, cardiovascular disease, and pulmonary restrictions. Compensation will include a summary of their highest peak power and mean power from the study.

5. **Briefly describe the research methodology. Attach copies of all test instruments/questionnaires that will be used.**

Design of the study: This study will utilize a pretest-posttest experimental design in which subjects will be randomly assigned on the first session to assume either a neutral spine or thoracic flexion position during recovery intervals. In the present study, thoracic flexion from T1 to T12 will be considered to be at an angle of 14° or more. The subjects will start with a five-minute warm-up at 75 W, followed by the first Wingate Anaerobic Test (WT_1), a four-minute active recovery interval pedaling at 1.1 W/kg, 90 revolutions per minute (rpm) in the assigned posture, the second Wingate Anaerobic Test (WT_2), and then a five-minute active recovery interval resting still and seated. After no less than a 48-hour period, he subjects will participate in

the second session where they will follow the same procedure, but perform the recovery intervals in the other thoracic position. Mean power and fatigue index will be measured during WT_1 and WT_2. Heart rate recovery will be measured during the first minute of both recovery intervals. VCO_2, and V_T will measured during passive recovery after WT_2.

Instrumentation:

HR will be recorded with a Polar heart rate monitor (Lake Success, NY). VCO_2 and V_T will be measured with a Parvomedics TrueOne Metabolic Cart (Sandy, UT). The Wingate Anaerobic Test will be performed on a Velotron cycler ergometer (Racer-Mate Inc., Seatlle, WA) for measurement of mean power and fatigue index. Thoracic flexion will be measured with inclinometers at T1 and T12 to assure real-time consistency of flexed and neutral positions (Van Blommestein, MaCrae, Lewis, & Morrissey, 2012). Reflective markers will be placed at T1, T7, T12, the iliac crest, the greater trochanter, and the lateral epicondyle of the femur. A 2-D Qualisys Motion Capture System (Göteborg, Sweden) will be set up to record spine and hip angles in the sagittal plane. One Qualysis ProReflex Motion Capture 240 Camera is used at a frequency of 240 Hz.

Measurement techniques and procedures:

There will be at least one experimenter who is CPR, first aid and AED certified present during all data collection. Thoracic position will be randomized by the flip of a coin for the first day of data collection. Subjects were either instructed to maintain a neutral spinal position or a flexed thoracic position during the exercise recovery portions. The neutral or control position will be held consistent and confirmed by inclinometers placed at T1 and T12. Subjects testing in

the flexed position will need to hold thoracic flexion at 14° or greater. A motion capture camera system will record the entire cycling session to record hip, trunk and thoracic angles.

Subjects will be instructed to wear tight-fitting black clothes. If this will be unavailable to them, garments will be provided by the lab. Reflective markers are positioned at T1, T7, T12, the iliac crest, the greater trochanter, and the lateral epicondyle of the femur. The ergometer set-up will be standardized to a cycling position for external validity and consistency. The distance from the top of the saddle to the pedal surface or seat height will be set to 100% of trochantric length (Nordeen-Snyder, 1977). The handlebar height will be adjusted to the same level as the seat height. Finally, handlebar fore-aft position will be modified to achieve a trunk angle of 20 to 30° (Ericson, Bratt, Nisell, Arborelius, & Ekholm, 1986; Korff, Newstead, Zandwijk, & Jensen, 2014).

Subjects will complete a five-minute warm-up at 75 W. Subjects will then complete WT_1 followed by a four-minute active recovery interval against resistance, 1.1 W/kg at 90 rpm in the thoracic posture selected for that session. Then, they will perform WT_2 at their usual self-selected thoracic flexion. For WT_1 and WT_2, the subjects will be instructed to pedal as fast as they could for a 30 second period at 0.075 kg per kg of body mass and will be given verbal encouragement.

Each subject will once again assume either thoracic flexion or a neutral spine and will be instructed to sit still on the ergometer in the laboratory for five minutes of active recovery with their hands on the ergometer handles. HRR, VCO_2, and V_T will all be recorded during the five-minute recovery data collection. HRR will be recorded for the first minute of recovery following both WT_1 and WT_2, as in similar research (Lamberts, Lemmink, Durandt, & Lambert, 2004; Lamberts, Swart, Noakes, & Lambert, 2008). VCO_2 and V_T will be recorded at one-minute

intervals during the passive recovery interval after WT_2. The metabolic cart will be used simultaneously to measure VCO_2 and tidal volume via a two-way breathing mouthpiece. These measures will be recorded every minute during the recovery period and the average will be used for analysis.

Data processing:

V_T and VCO_2 will be obtained through the metabolic cart at one-minute intervals for five minutes following WT_2. The average V_T and VCO_2 for each passive recovery period will be used for analysis. HRR will be calculated as the difference in HR from the end of each WT to one minute later. The MP, FI, , HRR, V_T, and VCO_2 data will be transferred into Excel (Microsoft Inc., Redmond, WA) for data analysis. Change in MP and FI from WT_1 to WT_2 will be calculated in Excel. FI will be calculated as [(max pedal speed – mean pedal speed)/max pedal speed ×100]. Subject data with less than 14° of thoracic flexion will be excluded from data processing.

Data analysis:

Dependent t-tests will be performed to compare FI, MP, HRR, V_T, and VCO_2 in neutral verses flexed thoracic recovery positions. Thoracic, trunk, and hip angle data will be reported for flexed and neutral intervals. Statistical significance will be established at $p < 0.05$. Bonferroni corrections will be applied for related variables. Effect size will be calculated. Cohen's d will be used to indicate the standardized difference between the two means. Data analysis will be performed with Excel (Microsoft Inc., Redmond, WA) and IBM SPSS 23 (Armonk, NY).

6. Give specific examples (with literature citations) for the use of your test instruments/questionnaires, or similar ones, in previous similar studies in your field.

The reliability of the Wingate test (WT) measured by the test-retest coefficient is good for peak power (P_{max}) ($r > 90$) and MP ($r > 0.91 - 0.93$) (Bar-Or, 1987; Neptune & Kautz, 2001; Patton, Murphy, & Frederick, 1985). Test-retest reliability for the WT tends to be higher for MP than P_{max} (Bar-Or, 1987). The WT conditions may also be relevant to other sport performances with intervals of high intensity exercise, like ice hockey, which has a similar fatigue curve in their skating tests (Cox, Miles, Verde, & Rhodes, 1995). The WT has been used in previous studies to examine exercise recovery (Dupont, Moalla, Matran, & Berthoin, 2007; Harbili, 2015; Lopez, Smoliga, & Zavorsky, 2014; Millar, Rakobowchuk, McCartney, & MacDonald, 2009). Repeated WTs are often used to compared recovery variables because the test is standardized and the intensity of the recovery interval can be quantified objectively (Lopez, Smoliga, & Zavorsky, 2014).

Dupont, Moalla, Matran, and Berthoin (2007) assessed the effects of different recovery intensities on the performance of two WTs. Subjects either recovered between repeated WTs passively, at 20% maximal aerobic power, or 40% maximal aerobic power. MP and P_{max} were significantly higher after a passive recovery interval (517±26 W and 1086±153 W, respectively) when compared to active recovery intervals at 20% (484±30 W and 973±112 W, respectively) and 40% (492±35 and 928±116 W, respectively). However, subjects only had a 15-second recovery interval. Other studies with a longer, four-minute recovery time between WTs found that active recovery leads to better performance (Lopez, Smoliga, & Zavorsky, 2014; Spierer et al., 2004). However, Lopez, Smoliga, and Zavorsky found that active recovery leads to 0.6 W/kg lower P_{max} only from the first to second WT.

Harbili (2015) examined the effect of recovery duration between repeated Wingate tests (WT) on $P_{max,}$ MP, and the FI of elite male cyclists. P_{max} significantly decreased in repeated WTs with recovery intervals of one (-70.42 W) and two minutes (-49.73 W), but did not significantly change with three-minute recovery intervals (-19.06 W). Mean power deceased in all recovery durations. The FI significantly decreased for one minute, but did not significantly change with two or three minute recovery intervals. The duration of recovery is a key factor in WT fatigue and P_{max} (Harbili, 2015). Type of recovery, whether passive or active, was not specified.

7. **Describe how your study design is appropriate to examine your question or specific hypothesis. Include a description of controls used, if any.**

The subjects will act as their own control by doing two testing days. One day they will recover with a neutral posture and the other with thoracic flexion. The differences in performance and recovery will be used to determine which posture may be more beneficial. Mean power and fatigue will be recorded during each maximal cycling test to determine changes in performance between postures. Respiratory and heart rate variables will be recorded during recovery to determine if posture affects recovery of the autonomic nervous and respiratory systems.

8. **Give specific examples (with literature citation) for the use of your study design, or similar ones, in previous similar studies in your field.**

A pretest-posttest experimental design has been used in many studies on exercise, power output, and cycling (Cheng et al., 2016; Engel et al., 2014; Engels, Kolokouri, Cieslak, & Wirth, 2001; Kimura et al., 2014; Lunn, Finn, & Axtell, 2009). Cheng et al. (2016) used this design to

examine the effects of caffeine on power and fatigue index. Engel et al., 2014 used a similar design to examine the hormonal changes in young athletes before and after a Wingate test. Engels, Kolokouri, Cieslak, and Wirth (2001) used a pretest-posttest design to measure the effects of ginseng on mean power and rate of fatigue. Kimura et al., (2014) used a similar design to test blood lactate before and after Wingate Anaerobic Tests. Another example would be a study done by Lunn et al. (2009) to measure the effect of cycle sprint training on body weight reduction.

9. Describe the potential risks to the human subjects involved.

Potential risks may include musculoskeletal injury, light-headedness, nausea, vomiting, chest pain, and/or fainting.

10. If the research involves potential risks, describe the safeguards that will be used to minimize such risks.

The researcher and research assistants will be ready to watch and listen for symptoms to prevent any kind of medical emergency. The cycle ergometer set-up previously mentioned has been chosen to minimize risk of musculoskeletal injury. Subjects will be asked to come hydrated and well-rested to the sessions. There will be at least one experimenter who is CPR, first aid and AED certified present during all data collection.

11. Describe how you will address privacy and/or confidentiality.

Subject data will be recorded with assigned numbers. Data will be kept on a hard drive protected via password.

Appendix C

Subject Demographics Data

Subject Demographics

Participant	Age (yrs)	Height (m)	Weight (kg)	Competitions in Last Year
1	31	1.70	74.84	1
2	28	1.80	83.01	3
3	23	1.78	62.60	17
4	26	1.83	74.84	10
5	27	1.70	63.50	18
6	24	1.78	80.29	26
7	29	1.80	74.84	5
8	41	1.83	76.20	10
9	35	1.83	79.38	6
10	35	1.88	78.93	14
11	37	1.80	85.73	3
12	44	1.78	73.94	6
13	21	1.73	73.94	4

Target Thoracic Flexion Position and Neutral Thoracic Flexion Positions

Participant	Flexion (degrees)	Neutral (degrees)
1	43	25
2	50	30
3	52	30
4	49	35
5	51	35
6	40	10
7	45	14
8	55	35
9	50	38
10	51	30
11	60	30
12	53	39
13	50	39

Appendix D

Mean Power and Fatigue Index

Flexed Condition Mean Power

	Mean Power (Watts)		
Participant	Sprint 1	Sprint 2	Change
1	587	576	-11
2	652	599	-53
3	600	537	-63
4	516	480	-36
5	550	511	-39
6	697	676	-21
7	566	558	-8
8	648	628	-20
9	637	599	-38
10	639	619	-20
11	679	641	-38
12	620	585	-35
13	587	583	-4

Neutral Condition Mean Power

	Mean Power (Watts)		
Participant	Sprint 1	Sprint 2	Change
1	545	527	-18
2	640	601	-39
3	525	490	-35
4	579	555	-24
5	558	521	-37
6	671	662	-9
7	564	548	-16
8	634	618	-16
9	609	591	-18
10	633	614	-19
11	684	606	-78
12	629	580	-49
13	589	584	-5

Flexed Condition Fatigue Index

	Fatigue Index (W/s)		
Participant	Sprint 1	Sprint 2	Change
1	6.8	8.1	1.3
2	7.3	1.7	-5.6
3	7.7	7.4	-0.3
4	3.4	13.2	9.8
5	7.7	7.7	0
6	6.1	3.3	-2.8
7	4.4	3.2	-1.2
8	8.7	9.9	1.2
9	9.3	6.9	-2.4
10	5.2	7.4	2.2
11	8.4	10.0	1.6
12	7.6	9.6	2.0
13	6.6	8.5	1.9

Neutral Condition Fatigue Index

	Fatigue Index (W/s)		
Participant	Sprint 1	Sprint 2	Change
1	7.1	8.1	1.0
2	4.8	5.5	0.7
3	5.7	5.7	0
4	6.5	5.4	-1.1
5	5.9	7.2	1.3
6	7.2	3.0	-4.2
7	4.3	4.3	0
8	10	10.1	0.1
9	6.1	5.0	-1.1
10	7.0	6.2	-0.8
11	9.0	3.8	-5.2
12	9.1	9.4	0.3
13	6.7	9.3	2.6

Appendix E

Tidal Volume, Volume of Carbon Dioxide, and Heart Rate Recovery

Flexed Condition: First Recovery Interval

	V_T (L)				VCO_2 (L/min)				HRR (bpm)
Participant	30s	90s	150s	210s	30s	90s	150s	210s	60s-0s
1	3.15	2.99	2.32	2.38	5.73	3.54	2.46	2.35	16
2	4.59	3.68	2.90	2.42	6.06	4.36	3.05	2.55	28
3	2.62	2.38	2.34	2.21	4.13	3.00	2.22	1.98	26
4	3.33	2.47	2.16	1.89	4.87	3.00	2.29	1.88	30
5	2.83	2.89	2.60	2.35	4.31	3.32	2.78	2.39	37
6	3.32	3.10	2.80	2.57	5.51	3.65	3.08	2.77	33
7	3.15	3.05	2.22	2.17	5.03	2.6	2.09	1.64	35
8	3.96	4.22	3.93	3.49	5.69	3.64	2.60	2.27	18
9	3.57	3.12	2.78	2.84	5.68	3.80	2.83	2.35	13
10	3.63	3.23	3.04	3.08	5.44	3.40	2.80	2.38	16
11	3.85	4.07	3.6	3.27	4.94	3.70	2.95	2.57	32
12	4.11	4.03	3.48	3.70	5.52	4.17	3.33	3.07	18
13	2.77	2.30	2.23	2.40	5.22	3.02	2.52	1.89	37

Flexed Condition: Second Recovery Interval

	V_T (L)				VCO_2 (L/min)				HRR (bpm)
Participant	30s	90s	150s	210s	30s	90s	150s	210s	60s-0s
1	3.46	2.66	2.44	2.42	5.78	3.61	2.45	2.19	16
2	4.31	3.13	2.61	2.44	5.17	3.37	2.33	2.34	20
3	2.89	2.29	2.6	2.09	3.63	2.49	1.77	1.97	16
4	3.39	2.71	2.25	2.12	4.54	2.8	2.34	2.12	28
5	2.77	2.68	2.26	2.02	4.28	3.03	2.32	1.88	15
6	3.36	3.04	2.82	2.55	5.06	3.76	2.99	2.54	24
7	3.30	2.49	2.12	2.13	4.30	2.51	2.06	1.73	40
8	4.13	4.00	3.99	3.61	4.55	3.23	2.26	2.25	16
9	3.92	3.16	2.74	2.58	5.05	3.18	2.21	2.26	16
10	3.46	3.50	3.01	2.7	4.35	3.05	2.40	2.16	15
11	3.57	3.65	3.51	3.41	4.48	3.09	2.60	2.36	21
12	3.60	3.89	3.75	3.37	4.54	3.76	2.93	2.69	12
13	2.90	2.46	2.34	2.28	4.69	3.01	2.44	2.14	34

Neutral Condition: First Recovery Interval

	V_T (L)				VCO_2 (L/min)				HRR (bpm)
Participant	30s	90s	150s	210s	30s	90s	150s	210s	60s-0s
1	3.71	3.50	3.1	2.63	7.08	4.15	2.87	2.37	24
2	4.43	2.90	2.8	2.49	5.78	3.84	2.91	2.75	22
3	2.74	2.22	2.02	2.06	4.01	2.61	2.15	2.12	29
4	2.82	2.76	2.4	2.23	4.36	3.23	2.40	2.29	19
5	2.80	2.71	2.35	2.26	3.38	2.74	2.39	2.09	38
6	3.77	3.44	3.01	2.95	5.40	3.89	3.27	2.77	27
7	3.54	3.28	2.45	2.57	5.12	3.00	2.21	2.11	31
8	4.03	4.09	4.00	3.44	5.74	3.65	2.83	2.35	16
9	3.78	3.09	2.75	2.50	5.48	3.92	2.66	2.20	13
10	3.85	3.51	3.27	3.41	5.20	3.78	3.04	2.20	23
11	4.08	4.09	4.00	3.67	5.26	3.73	2.86	2.62	32
12	4.08	4.21	4.04	3.91	5.45	3.74	3.24	3.00	19
13	2.71	2.64	2.34	2.09	4.89	2.92	2.32	2.23	29

Neutral Condition: Second Recovery Interval

	V_T (L)				VCO_2 (L/min)				HRR (bpm)
Participant	30s	90s	150s	210s	30s	90s	150s	210s	60s-0s
1	3.5	3.07	2.78	2.54	5.47	3.67	2.71	2.24	19
2	3.88	3.02	2.41	2.48	4.96	3.26	2.33	2.12	17
3	2.76	2.48	2.21	2.31	3.31	2.49	2.07	1.93	22
4	3.38	2.61	2.23	2.26	4.68	2.65	2.2	1.57	17
5	2.71	2.46	2.25	2.17	4.08	2.57	1.99	1.93	15
6	3.7	3.22	2.96	2.82	4.88	3.78	3.02	2.58	20
7	3.69	3.04	2.81	2.07	4.62	2.64	2.13	1.5	30
8	3.96	4.24	3.58	3.36	4.74	2.98	2.32	2.11	16
9	3.63	3.07	2.66	2.46	4.84	2.91	2.2	2.01	14
10	3.72	3.44	3.07	2.79	4.08	3.23	2.44	2.16	25
11	3.81	3.67	3.52	2.95	4.23	3.22	2.46	2.2	17
12	3.98	4.21	3.99	3.65	5.15	3.8	3.05	2.63	16
13	2.8	2.9	2.32	2.28	3.81	3.46	2.65	2.22	32

Flexed Condition: Average Between Both Recovery Intervals

Participant	Average V_T (L)	Average VCO_2 (L/min)	Average HRR (bpm)
1	2.72750	3.52000	16.0
2	3.26000	3.75625	24.0
3	2.42750	3.06750	21.0
4	2.54000	2.73750	29.0
5	2.55000	3.07500	26.0
6	2.94500	3.31500	28.5
7	2.57875	3.21375	37.5
8	3.91625	3.10000	17.0
9	3.08875	3.36875	14.5
10	3.20625	3.34000	15.5
11	3.61625	3.26500	26.5
12	3.74125	3.57750	15.0
13	2.46000	3.32125	35.5

Neutral Condition: Average Between Both Recovery Intervals

Participant	Average V_T (L)	Average VCO_2 (L/min)	Average HRR (bpm)
1	3.10375	3.82000	21.5
2	3.05125	3.49375	19.5
3	2.35000	2.58625	25.5
4	2.58625	2.92250	18.0
5	2.46375	2.64625	26.5
6	3.23375	3.69875	23.5
7	2.93125	2.91625	30.5
8	3.83750	3.34000	16.0
9	2.99250	3.27750	13.5
10	3.38250	3.26625	24.0
11	3.72375	3.32250	24.5
12	4.00875	3.75750	17.5
13	2.51000	3.06250	30.5

Appendix F

Thoracic Flexion

Flexed Thoracic Flexion

	Average Thoracic Flexion (deg)	
Participant	Recovery 1	Recovery 2
1	36.6	36.8
2	50.3	47.4
3	54.7	59.9
4	48.8	49.0
5	35.0	35.1
6	33.7	34.2
7	39.3	37.7
8	50.3	50.2
9	47.3	46.4
10	48.9	49.7
11	53.3	52.2
12	44.0	45.3
13	49.8	49.8

Neutral Thoracic Position

	Average Thoracic Flexion (deg)	
Participant	Recovery 1	Recovery 2
1	21.8	23.4
2	33.6	29.1
3	41.3	40.6
4	39.2	39.6
5	30.7	31.2
6	14.0	13.6
7	25.9	24.9
8	31.0	33.2
9	38.0	36.8
10	29.1	61.8
11	29.2	30.7
12	31.2	31.1
13	38.4	36.2

Appendix G

Statistics

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	TF NC	32.1231	13	8.36911	2.32117
	TF FC	45.6231	13	7.32111	2.03051
Pair 2	HRR NC	21.2692	13	4.97751	1.38051
	HRR FC	23.5385	13	7.81415	2.16725
Pair 3	V_T NC	3.1925	13	.54489	.15112
	V_T FC	3.0044	13	.51435	.14266
Pair 4	VCO_2 NC	3.2606	13	.39665	.11001
	VCO_2 FC	3.2813	13	.25913	.07187
Pair 5	ΔMP NC	-28.7692	13	18.87306	5.23444
	ΔMP FC	-29.6923	13	17.53275	4.86271
Pair 6	ΔFI NC	-.6846	13	1.93513	.53671
	ΔFI FC	.5923	13	3.60681	1.00035

Cohen's d and Effect Size

		Cohen's d	Effect Size r
Pair 1	TF NC & TF FC	1.71	0.65
Pair 2	HRR NC & HRR FC	0.33	0.17
Pair 3	V_T NC & V_T FC	0.36	0.17
Pair 4	VCO_2 NC & VCO_2 FC	0.062	0.031
Pair 5	ΔMP NC & MP FC	0.051	0.025
Pair 6	ΔFI NC & FI FC	0.44	0.22

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	TF	-13.50000	5.71110	1.58397	-16.9511	-10.0488	-8.523	12	.000
Pair 2	HRR	-2.26923	7.43756	2.06281	-6.76370	2.22524	-1.100	12	.293
Pair 3	V_T	.18808	.40576	.11254	-.05712	.43327	1.671	12	.121
Pair 4	VCO_2	-.02077	.28079	.07788	-.19045	.14891	-.267	12	.794
Pair 5	ΔMP	.92308	17.63265	4.89042	-9.73223	11.57838	.189	12	.853
Pair 6	ΔFI	-1.27692	4.15174	1.15148	-3.78579	1.23195	-1.109	12	.289